Escherichia coli O157

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Escherichia coli O157:H7 (O157) are noninvasive and weak biofilm producers; however, a subset of O157 are exceptions. O157 ATCC 43895 forms biofilms and invades epithelial cells. Tn*5* mutagenesis identified mutation insertions that map within the curli *csgB* fimbriae locus to be responsible for both phenotypes. Screening of O157 strains for biofilm formation and cell invasion identify a bovine and a clinical isolate with those characteristics. A single base pair A to T transversion, intergenic to the curli divergent operons *csgDEFG* and *csgBAC,* is present only in biofilm-producing and invasive strains. Using site-directed mutagenesis, this single base change was introduced into two curli-negative/noninvasive O157 strains and modified strains to form biofilms, produce curli, and gain invasive capability. Transmission electron microscopy (EM) and immuno-EM confirmed curli fibers. EM of bovine epithelial cells (MAC-T) co-cultured with curli-expressing O157 show intracellular bacteria. The role of curli in O157 persistence in cattle was examined by challenging cattle with curli-positive and -negative O157 and comparing carriage. The duration of bovine colonization with the O157 curli-negative mutant was shorter than its curli-positive isogenic parent. These findings definitively demonstrate that a single base pair stably confers biofilm formation, epithelial cell invasion, and persistence in cattle.

Keywords: E. coli O157 ; cattle ; curli ; biofilm ; cell invasion

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Enterohemorrhagic Escherichia coli (EHEC) cause human disease with symptoms ranging from self-limited watery diarrhea to life-threatening hemorrhagic colitis, and hemolytic uremic syndrome [1][2]. E. coli O157:H7 (O157) is the best studied EHEC serotype and is the predominant strain associated with disease outbreaks in North America, the United Kingdom, and Japan [3][4][5]. Cattle and other ruminants carry this pathogen with no apparent symptoms [6][7] and are the most common source for human infections [8][9]. O157 colonize at the bovine recto-anal junction (RAJ) and the bacteria persist in the feces of individual animals from a few days to several months [10][11]. Attachment to biological surfaces is a first critical step in colonization and is mediated by multiple bacterial factors. Surface-associated factors of O157 contributing to tissue adherence and persistence in the bovine host include O-antigen [12], fimbriae [13], adhesins such as intimin [14], and some autotransporters [15]. There is evidence that the duration of colonization and the bovine immune responses are strain/variant dependent [16][17].

Curli fimbriae, comprised of polymerized amyloid protein, are expressed on the surface of many members of the Enterobacteriaceae and other Gammaproteobacteria [18]. Curli binds amyloid-specific dyes, such as Congo red and certain host proteins, including fibronectin, laminin, and plasminogen [19][20]. During infections, curli complexes with extracellular matrix DNA. In a mouse model for lupus erythematosus autoimmunity, these curli-DNA complexes interact with Toll-like receptors (TLRs) 2 and 9 on dendritic and macrophage cells resulting in the production of autoantibodies [21]. In most non-O157 E. coli, curli is regulated by σs and synthesized at low temperature, in nutrient-deprivation, and/or in stationary phase, conditions that promote biofilm formation [22]. Curli synthesis requires the expression of genes from two divergently transcribed operons, designated csgDEFG and csgBAC. Genes with identified functions include the regulator CsgD, the type VIII secretion machinery components CsgE-G, the curli major subunit CsgA, and the curli nucleation protein CsgB [23][24]. The intergenic region between csgDEFG and csgBAC is large and contains many putative binding sites for regulatory factors. CsgD is essential for the transcription of the curli operons [19]. Curli promotes biofilm adhesion to abiotic surfaces as well as to mammalian cells [25][26][27][28]. Although both operons are present in all sequenced O157 strains [29][30][31], the majority of O157 (approximately 95%) are curli-negative. This is because the prophage carrying Shiga toxin type-1 inserts into mlrA, a positive regulator of csgD [32]. The few curli-positive O157 strains produce curli constitutively, including at 37 °C, and have acquired a suppressor mutation overriding the normal requirement for mlrA [33][34].

O157 is a weak biofilm producer and is considered an extracellular pathogen [35][36]. However, some strains do not meet this general characterization. In a previous study, we showed that O157 strain 43895, an outbreak isolate from hamburger, produces biofilms at 37 °C, invades epithelial cells, and persists longer in cattle than a biofilm-negative strain [16]. Curli expression has been found in certain O157 strains [34], but the underlying mechanism has not been fully explored.

References

1. Paton, J.C.; Paton, A.W. Pathogenesis and diagnosis of Shiga toxin-producing Escherichia coli infections. Clin. Microbiol. Rev. 1998, 11, 450–479. [Google Scholar] [CrossRef] [PubMed]
2. Karmali, M.A.; Steele, B.T.; Petric, M.; Lim, C. Sporadic cases of haemolytic-uraemic syndrome associated with faecal cytotoxin and cytotoxin-producing Escherichia coli in stools. Lancet 1983, 1, 619–620. [Google Scholar] [CrossRef]
3. Griffin, P.M.; Tauxe, R.V. The epidemiology of infections caused by Escherichia coli O157:H7, other enterohemorrhagic E. coli, and the associated hemolytic uremic syndrome. Epidemiol. Rev. 1991, 13, 60–98. [Google Scholar] [CrossRef] [PubMed]
4. Michino, H.; Araki, K.; Minami, S.; Takaya, S.; Sakai, N.; Miyazaki, M.; Ono, A.; Yanagawa, H. Massive outbreak of Escherichia coli O157:H7 infection in schoolchildren in Sakai City, Japan, associated with consumption of white radish sprouts. Am. J. Epidemiol. 1999, 150, 787–796. [Google Scholar] [CrossRef]
5. Smith, H.R. Escherichia coli O157 infections: The Scottish experience. Hosp. Med. 1998, 59, 164. [Google Scholar]
6. Blanco, M.; Blanco, J.E.; Blanco, J.; Gonzalez, E.A.; Mora, A.; Prado, C.; Fernández, L.; Rio, M.; Ramos, J.; Alonso, M.P. Prevalence and characteristics of Escherichia coli serotype O157:H7 and other verotoxin-producing E. coli in healthy cattle. Epidemiol. Infect. 1996, 117, 251–257. [Google Scholar] [CrossRef]
7. Sheng, H.; Davis, M.A.; Knecht, H.J.; Hancock, D.D.; Donkersgoed, J.V.; Hovde, C.J. Characterization of a Shiga toxin-, intimin-, and enterotoxin hemolysin-producing Escherichia coli ONT:H25 strain commonly isolated from healthy cattle. J. Clin. Microbiol. 2005, 43, 3213–3220. [Google Scholar] [CrossRef]
8. Hancock, D.D.; Besser, T.E.; Kinsel, M.L.; Tarr, P.I.; Rice, D.H.; Paros, M.G. The prevalence of Escherichia coli O157.H7 in dairy and beef cattle in Washington State. Epidemiol. Infect. 1994, 113, 199–207. [Google Scholar] [CrossRef]
9. Zhao, T.; Doyle, M.P.; Shere, J.; Garber, L. Prevalence of enterohemorrhagic Escherichia coli O157:H7 in a survey of dairy herds. Appl. Environ. Microbiol. 1995, 61, 1290–1293. [Google Scholar] [CrossRef]
10. Naylor, S.W.; Low, J.C.; Besser, T.E.; Mahajan, A.; Gunn, G.J.; Pearce, M.C.; McKendrick, I.J.; Smith, D.E.; Gallyet, D.L. Lymphoid Follicle-Dense Mucosa at the Terminal Rectum is the Principal Site of Colonization of Enterohemorrhagic Escherichia coli O157:H7 in the Bovine Host. Infect. Immun. 2003, 71, 1505–1512. [Google Scholar] [CrossRef]
11. Sheng, H.; Davis, M.A.; Knecht, H.J.; Hovde, C.J. Rectal administration of Escherichia coli O157:H7: Novel model for colonization of ruminants. Appl. Environ. Microbiol. 2004, 70, 4588–4595. [Google Scholar] [CrossRef] [PubMed]
12. Sheng, H.; Lim, J.Y.; Watkins, M.K.; Minnich, S.A.; Hovde, C.J. Characterization of an Escherichia coli O157:H7 O-antigen deletion mutant and effect of the deletion on bacterial persistence in the mouse intestine and colonization at the bovine terminal rectal mucosa. Appl. Environ. Microbiol. 2008, 74, 5015–5022. [Google Scholar] [CrossRef] [PubMed]
13. Vogeleer, P.; Tremblay, Y.D.N.; Jubelin, G.; Jacques, M.; Harel, J. Biofilm-forming abilities of Shiga toxin-producing Escherichia coli isolates associated with human infections. Appl. Environ. Microbiol. 2015, 82, 1448–1558. [Google Scholar] [CrossRef] [PubMed]
14. Lloyd, S.J.; Ritchie, J.M.; Torres, A.G. Fimbriation and curliation in Escherichia coli O157:H7: A paradigm of intestinal and environmental colonization. Gut Microbes 2012, 3, 272–276. [Google Scholar] [CrossRef] [PubMed]
15. Wells, T.J.; Sherlock, O.; Rivas, L.; Mahajan, A.; Beatson, S.A.; Torpdahl, M.; Webb, R.I.; Allsopp, L.P.; Gobius, K.S.; Gally, D.L.; et al. EhaA is a novel autotransporter protein of enterohemorrhagic Escherichia coli O157:H7 that contributes to adhesion and biofilm formation. Environ. Microbiol. 2008, 10, 589–604. [Google Scholar] [CrossRef]
16. Sheng, H.; Wang, J.; Lim, J.Y.; Davitt, C.; Minnich, S.A.; Hovde, C.J. Internalization of Escherichia coli O157:H7 by bovine rectal epithelial cells. Front. Microbiol. 2011, 2, 32. [Google Scholar] [CrossRef]
17. Corbishley, A.; Ahmad, N.I.; Hughes, K.; Hutchings, M.R.; McAteer, S.P.; Connelley, T.K.; Helen Brown, H.; Gally, D.L.; Tom, N.; McNeilly, T.N. Strain-dependent cellular immune responses in cattle following Escherichia coli O157:H7 colonization. Infect. Immun. 2014, 82, 5117–5131. [Google Scholar] [CrossRef]
18. Smith, D.R.; Price, J.E.; Burby, P.E.; Blanco, L.P.; Chamberlain, J.; Chapman, M.R. The production of curli amyloid fibers is deeply integrated into the biology of Escherichia coli. Biomolecules 2017, 7, 75. [Google Scholar] [CrossRef]
19. Barnhart, M.M.; Chapman, M.R. Curli biogenesis and function. Annu. Rev. Microbiol. 2006, 60, 131–147. [Google Scholar] [CrossRef]
20. Olsen, A.; Wick, M.J.; Morgelin, M.; Bjorck, L. Curli, fibrous surface proteins of Escherichia coli, interact with major histocompatibility complex class I molecules. Infect. Immun. 1998, 66, 944–949. [Google Scholar] [CrossRef]
21. Tursi, S.A.; Lee, E.Y.; Medeiros, N.J.; Lee, M.H.; Nicastro, L.K.; Buttaro, B.; Gallucci, S.; Wilson, R.P.; Wong, G.C.L.; Tukel, C. Bacterial amyloid curli acts as a carrier for DNA to elicit an autoimmune response via TLR2 and TLR9. PLoS Pat

hog. 2017, 13. [Google Scholar] [CrossRef] [PubMed]

22. Olsen, A.; Arnqvist, A.; Hammar, M.; Sukupolvi, S.; Normark, S. The RpoS sigma factor relieves H-NS-mediated transcr iptional repression of csgA, the subunit gene of fibronectinbinding curli in Escherichia coli. Mol. Microbiol. 1993, 7, 523–536. [Google Scholar] [CrossRef] [PubMed]
23. Chapman, M.R.; Robinson, L.S.; Pinkner, J.S.; Roth, R.; Heuser, J.; Hammar, M.; Normark, S.; Hultgren, S.J. Role of E scherichia coli curli operons in directing amyloid fiber formation. Science 2002, 295, 851–855. [Google Scholar] [Cross Ref] [PubMed]
24. Hammar, M.; Arnqvist, A.; Bian, Z.; Olsen, A.; Normark, S. Expression of two csg operons is required for production of fi bronectin- and congo red-binding curli polymers in Escherichia coli K-12. Mol. Microbiol. 1995, 18, 661–670. [Google S cholar] [CrossRef]
25. Kikuchi, T.; Mizunoe, Y.; Takade, A.; Naito, S.; Yoshida, S. Curli fibers are required for development of biofilm architectu re in Escherichia coli K-12 and enhance bacterial adherence to human uroepithelial cells. Microbiol. Immunol. 2005, 4 9, 875–884. [Google Scholar] [CrossRef]
26. Uhlich, G.A.; Cooke, P.H.; Solomon, E.B. Analyses of the red-dry-rough phenotype of an Escherichia coli O157:H7 strai n and its role in biofilm formation and resistance to antibacterial agents. Appl. Environ. Microbiol. 2006, 72, 2564–2572. [Google Scholar] [CrossRef]
27. Vidal, O.; Longin, R.; Prigent-Combaret, C.; Dorel, C.; Hooreman, M.; Lejeune, P. Isolation of an Escherichia coli K-12 mutant strain able to form biofilms on inert surfaces: Involvement of a new ompR allele that increases curli expression. J. Bacteriol. 1998, 180, 2442–2449. [Google Scholar] [CrossRef]
28. Cookson, A.L.; Cooley, W.A.; Woodward, M.J. The role of type 1 and curli fimbriae of Shiga toxin-producing Escherichia coli in adherence to abiotic surfaces. Int. J. Med. Microbiol. 2002, 292, 195–205. [Google Scholar] [CrossRef]
29. Perna, N.T.; Plunkett, G.; Burland, V.; Mau, B.; Glasner, J.D.; Rose, D.J.J.; Mayhew, G.F.; Evans, P.S.; Gregor, J.; Kirkp atrick, H.A.; et al. Genome sequence of enterohaemorrhagic Escherichia coli O157:H7. Nature 2001, 409, 529–533. [G oogle Scholar] [CrossRef]
30. Hayashi, T.; Makino, K.; Ohnishi, M.; Kurokawa, K.; Ishii, K.; Yokoyama, K.; Han, C.G.; Ohtsubo, E.; Nakayama, K.; Mu rata, T.; et al. Complete genome sequence of enterohemorrhagic Escherichia coli O157:H7 and genomic comparison w ith a laboratory strain K-12. DNA Res. 2001, 8, 11–22. [Google Scholar] [CrossRef]
31. Sheng, H.; Duan, M.; Hunter, S.S.; Minnich, S.A.; Settles, M.L.; New, D.D.; Chase, J.R.; Fagnan, M.W.; Hovde, C.J. Hig h-Quality Complete Genome Sequences of Three Bovine Shiga Toxin-Producing Escherichia coli O177:H- (fliCH25) Iso lates harboring virulent stx2 and multiple plasmids. Genome Announc. 2018, 6. [Google Scholar] [CrossRef] [PubMed]
32. Uhlich, G.A.; Chen, C.Y.; Cottrell, B.J.; Hofmann, C.S.; Yan, X.; Nguyen, L. Stx1 prophage excision in Escherichia coli s train PA20 confers strong curli and biofilm formation by restoring native mlrA. FEMS Microbiol. Lett. 2016, 363. [Google Scholar] [CrossRef] [PubMed]
33. Bian, Z.; Brauner, A.; Li, Y.; Normark, S. Expression of and cytokine activation by Escherichia coli curli fibers in human sepsis. J. Infect. Dis. 2000, 181, 602–612. [Google Scholar] [CrossRef]
34. Uhlich, G.A.; Keen, J.E.; Elder, R.O. Mutations in the csgD promoter associated with variations in curli expression in ce rtain strains of Escherichia coli O157:H7. Appl. Environ. Microbiol. 2001, 67, 2367–2370. [Google Scholar] [CrossRef]
35. Luck, S.N.; Bennett-Wood, V.; Poon, R.; Robins-Browne, R.M.; Hartland, E.L. Invasion of epithelial cells by locus of ent erocyte effacement-negative enterohemorrhagic Escherichia coli. Infect. Immun. 2005, 73, 3063–3071. [Google Schola r] [CrossRef]
36. Lim, J.Y.; La, H.J.; Sheng, H.; Forney, L.J.; Hovde, C.J. Influence of plasmid pO157 on Escherichia coli O157:H7 Sakai biofilm formation. Appl. Environ. Microbiol. 2010, 76, 963–966.

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